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Original research

Atmospheric pressure and infra-renal abdominal aortic aneurysm rupture: A single observational study and a comprehensive review of literature

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ABSTRACT

Introduction: There have been various suggestions that abdominal aortic aneurysm rupture (rAAA) might have a seasonal variation depending on atmospheric pressure (AP) alteration. Despite above suggestions no study has yet examined the effect of fluctuation in AP on daily, seasonal, monthly, AAA size and co-morbidities to reach a conclusive outcome.

Methods: A total of fifty ($n = 50$) ruptured AAA over a 5-year period were retrospectively studied. Local meteorological data on AP were obtained from UK Meteorological Office. The data was subjected to statistical analysis using Student's *t*-test, linear regression (Pearson correlation Coefficient) and Coefficient of determination to establish any casual link between AP and incidences of rAAA on daily, seasonal and monthly basis. The casual link was also assessed between AP, AAA size and co-morbidities. The outcome is presented in a format of comprehensive review of literature that detected only 6 papers in MEDLINE and EMBASE from 1951 to 2012 in UK.

Conclusion: There appears to be a significant correlation between mean monthly pressures and mean monthly rupture incidence (Pearson)($n = 12$; $r = -0.61$; $p < 0.034$; rsq = 0.37). The periods of low AP are associated with higher incidence of rupture (rAAA $n = 29$ at mean atmospheric pressure 1012 mB Vs rAAA $n = 12$ at mean atmospheric pressure 1016 mB Vs rAAA $n = 9$ at mean atmospheric pressure of 1020 mB) in our study and all reviewed literature. In addition, no casual link between AP to co-morbidities (diabetes mellitus, hypertension ischaemic heart disease, chronic obstructive pulmonary disease) and AAA size could be established or found in the literature.

1. Introduction

To date numerous studies have been conducted to understand the aetiology, patho-physiology and the course of abdominal aortic aneurysm (AAA) and its path to rupture.[1] AAA has a complex aetiology but it has been speculated that familial and degenerative components play a major role. Despite recognition of certain modifiable risk factors, such as hypertension, smoking and chronic obstructive pulmonary disease, predicting AAA rupture remains elusive to this date.[2,3] It has been suggested that seasonal variation of meteorological parameters might be associated with cardiovascular mortality.[4] This correlation is distinctively supported in the literature with regards to ischaemic coronary artery disease in the months of winter and summer.[5] However, the influence of such factors (meteorological parameters) with emphasis on atmospheric pressure and its correlation to infra-renal AAA rupture incidence in the literature is vague and inconclusive. Therefore, the objective of this study is to establish whether there is a correlation between atmospheric pressure (AP), and the incidences of infra-renal AAA rupture on a daily, monthly and seasonal basis. Similarly the size of AAA and its correlation to daily, monthly and seasonal atmospheric pressure will be assessed in synergy. The outcome will be presented in conjunction with other studies to reach a possible conclusive outcome.

2. Patients and methods

A retrospective study of all ruptured infra-renal AAA from November 2007 to June 2012 at Mid Essex Hospital Services NHS Trust with estimated population coverage of 360,000 was designed. The hospital database for all ruptured cases that were operated upon was searched and a total of fifty-two ($n = 52$) patients were identified. Two patients ($n = 2$) presented with iliac aneurysm rupture and were thus excluded from the study. None of the patients were on any local screening programme. All patient records ($n = 50$) were recruited and studied. The data on age, sex and co-morbidities (Diabetes mellitus (DM), Hypertension (HTN), Ischaemic heart disease (IHD), chronic obstructive pulmonary disease (COPD), smoking

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Table 1.1
Patients demographics.

Number of patients	$n = 50$
Mean age	75.4 (Range, 50–90 years)
Male to female ratio (8:1)	$n = 42/50$ (84% male Vs 16%)
Hypertensive	$n = 28/50$ (56%)
Ischaemic heart disease	$n = 14/50$ (28%)
Chronic obstructive pulmonary disease	$n = 4/50$ (8%)
Active smoker	$n = 12/50$ (24%)
Diabetes mellitus	$n = 2/50$ (4%)
Male patients	$n = 42$
Mean age	74 (Range, 50–90 years)
Hypertensive	$n = 22/42$ (52%)
Ischaemic heart disease	$n = 13/42$ (30.9%)
Chronic obstructive pulmonary disease	$n = 4/42$ (9.5%)
Active smoker	$n = 11/42$ (26.1%)
Diabetes mellitus	$n = 2/42$ (4.7%)
Female patients	$n = 8$
Mean age	80.7 (Range, 62–88 years)
Hypertensive	$n = 6/8$ (75%)
Ischaemic heart disease	$n = 1/8$ (12.8%)
Chronic obstructive pulmonary disease	$n = 0/8$ (0%)
Active smoker	$n = 1/8$ (12.8%)
Diabetes mellitus	$n = 0/8$ (0%)

history) were collected (Table 1.1). Similarly, the exact aneurysm size for each patient was calculated. This was achieved by examination of all individual computed tomography angiogram (CTA) or the computed tomography (CT). The method of measurement was antero-posterior external diameter on true longitudinal plane in millimetres. The exact date for all ruptures and the preceding day was sent to the regional Meteorological Office (UK). The mean daily atmospheric pressure in Millibars (mB) on both days for all patients was received ($n = 100$). The data was subjected to statistical analysis using AcaStat software Version 8.1.11(2012). Student's *t*-test, linear regression (Pearson correlation Coefficient) and Coefficient of determination were utilized as a part of the statistical analysis and a *p*-value of <0.05 was taken to reflect significance. Total ruptures were expressed cumulatively per month and seasonally as defined by UK Meteorological office (spring, March–May; Summer, June–August; autumn, September–November; winter, December–February). Due to the retrospective nature of the study, lack of any intervention, availability of the essential data for clinical reasons and possible service improvement no ethical approval was found necessary.

An electronic search of MEDLINE and EMBASE for any literature in English language from 1951 to May 2012 was performed. The references were also reviewed for any additional studies. This search was limited to any study on atmospheric pressure (AP) and abdominal aortic aneurysm (AAA). To the best of our knowledge we found a total of 10 published papers. All papers were retrieved and studied. In our review, we included only those papers ($n = 6$) (Table 1.2) that were conducted in UK. This is due to the significant climate difference to Australian, Mediterranean and North American countries ($n = 4$).

3. Results

A total of fifty ($n = 50$) ruptured AAA cases were identified during this period. The male-to-female ratio was 7:1 with mean age of 75.4 (range, 50–90 years). The most common co-morbidity was hypertension (HTN) (56.0%)($n = 28$) followed by ischaemic heart disease (IHD) (28%)($n = 14$), chronic obstructive pulmonary disease (COPD) (8.0%)($n = 4$) and diabetes mellitus (DM) (4.0%)($n = 2$). Only 24.0% were ($n = 12$) were found to be active smoker at the time of the operation (Table 1.1) The mean AAA rupture size was 72.52 mm (range, 40–100 mm). The highest number of ruptures occurred in the month of March ($n = 7$; Mean pressure = 1015.40 mB) and June ($n = 7$; Mean pressure = 1015.24 mB) followed by February ($n = 6$; Mean pressure = 1004.68 mB) and April ($n = 6$; Mean pressure: 1007.73 mB) with lowest in August ($n = 1$: Mean pressure = 1018.2 mB). In terms of seasonal rupture, spring had the highest incidence of rupture ($n = 17$; Mean pressure: 1012.21 mB) with least occurring in autumn ($n = 9$; Mean pressure: 1020.23 mB).

4. Atmospheric pressure and seasonal variations

The data was analysed in a number of ways to demonstrate any casual relationships between mean daily atmospheric (on day of rupture), mean pressure difference (day before and on day of rupture), aneurysm size and rupture presentation. The *p* value ($p \geq 0.24$) (Student's *t*-test) for pressure difference on day basis was (the day of rupture and its preceding day) insignificant. However, The *p* value (Student's *t*-test) for mean monthly and mean seasonal pressure difference showed boarder line correlation respectively ($p = 0.059$, mean monthly pressure difference = 2.08 mB; range, −2.51–10.85 mB; 95% CI ± 2.93), ($p = 0.054$, mean seasonal pressure difference = 1.65 mB; range, 0.19–3.41 mB; 95% CI ± 0.10). There was a significant correlation between mean monthly pressure and mean monthly rupture incidence (Pearson)($n = 12$; $r = -0.61$; $p < 0.034$; rsq = 0.37). No casual relationship was detected between mean seasonal pressure and mean seasonal rupture frequencies (Pearson)($n = 4$; $r = -0.108$; $p < 0.89$; rsq = 0.0118). This was also insignificant for monthly and seasonal pressure differences to the rupture incidences.

5. Aneurysm size and co-morbidities

The data analysis failed to exhibit any correlation between the mean daily, monthly and seasonal pressure and that of the aneurysm size in all categories (Pearson and Pearson correlation Coefficient) ($p < 0.58$, $r = -0.078$, rsq = 0.006; $p < 0.29$, $r = 0.330$, rsq = 0.109; $p < 0.66$, $r = 0.331$, rsq = 0.109). Further sub-analysis of the co-morbidities (Diabetes mellitus, Hypertension, Ischaemic heart disease, chronic obstructive pulmonary disease, smoking history) did not detect any casual relation to monthly and seasonal rupture incidences.

Table 1.2
The included studies ($n = 6$).

Study/Year	Cases	Daily	Monthly	Seasonal	Comments
Talbot et al. (1972)	$n = 165$	Not assessed.	• Peak rupture in December ($n = 27$).	• Peak rupture in Winter.	• No AP recorded. • No *p* value given.
Varty et al. (1995)	$n = 372$	Not assessed.	• Peak rupture March ($n = 44$). • No correlation ($p = 0.96$).	• No statistical correlation.	• No AP records provided.
Ballaro et al. (1998)	$n = 19,599$	Not assessed.	• Peak rupture in December, January ($p = 0.003$).	• Peak rupture in winter ($p = 0.01$).	• No AP records provided.
Bown et al. (2003)	$n = 223$	Not significant ($p = 0.15$).	• Peak rupture in December ($p = 0.05$).	• No seasonal comparison assessed.	• Positive correlation between rupture & proceeding months mean AP ($p = 0.01$).
Harkin et al. (2005)	$n = 144$	Positive correlation ($p > 0.029$).	• Peak rupture in April & September ($p < 0.03$).	• Peak rupture in Autumn & Spring.	• Mean AP was lower on days of rupture.
Smith et al. (2008)	$n = 182$	Lower AP on days of rupture ($p = 0.025$).	Not significant ($p = 0.079$).	• Not assessed.	• Low AP associated with increased risk of rupture.

Table 1.3
Seasonal correlation of Co-morbidities.

Season	Ruptures	HTN	IHD	Smoker	COPD	DM
Spring	17	7	3	2	1	0
Summer	12	8	5	3	1	0
Autumn	9	7	1	1	0	0
Winter	12	6	5	6	2	2

6. Discussion

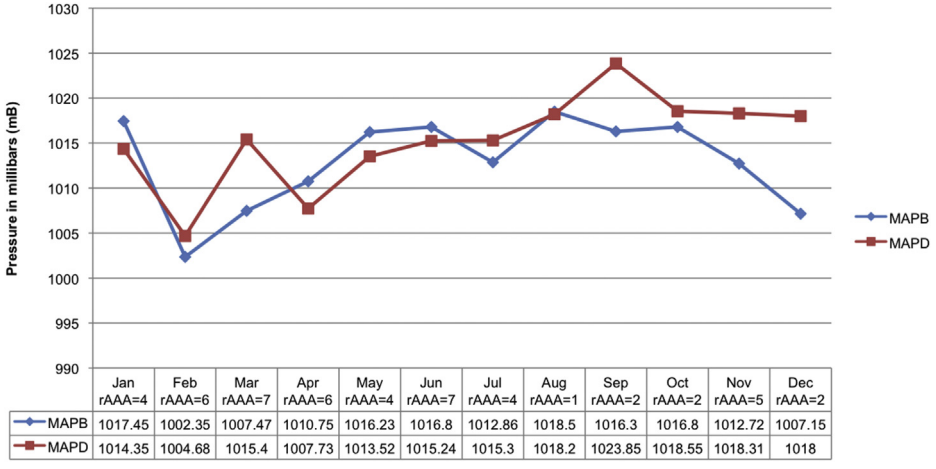
The "Laplace law" has been traditionally used to ascribe an infra-renal AAA deformation and its rupture. According to this law there is a direct relationship between tension, pressure and the radius of each cylindrical structure. In simple terms the pressure gradient across the aortic wall is dependent on intra and extra luminal pressures.[6] The intra-luminal pressure is produced by a discrete quanta of energy through each cardiac cycle within the circulating blood and its compositions. This results in oscillation, loading and unloading of the fibres in the aneurysm wall and produces changes in diameter of the vessel wall.[7] Due to a relative high concentration of collagen and elastin within abdominal aorta, considerable dispensability is permitted, but once the gradient pressure exceeds 100 mmHg (133 mB) the aorta becomes very stiff, and recruitment of non-distensible collagen occurs.[7] The extra-luminal pressure is referred to the intra abdominal and tissue pressure that measures around 5–7 mmHg (6.66–9.33 mB) and can rise to acceptable levels of 12 mmHg (16 mB).[8]

The alteration in AP has been shown to affect the partial pressure of the oxygen (*P*O2) and carbon dioxide (*P*CO2) in circulatory blood. It appears that the effect is considerably significant and averages to about 2.3% per 100 mmHg (133 mB) for partial pressure of oxygen and 1.0% per 100 mmHg (133 mB) for carbon dioxide. Similarly, it has been calculated that 3.7% of total variation in *P*O2 is attributed to AP alterations.[9] In addition, it has been suggested that seasonal meteorological parameters can influence blood pressure in hypertensive and normotensive indididuals.[10,11] The combination of hypoxia, hypercarbia and alteration in blood pressure secondary to AP fluctuations can result in notable circulatory disturbances. These include activation of chemoreceptors, arterial and non-arterial baroreceptors for increase in sympathetic stimulation thus secretion of adrenaline and noradrenaline.[12,13] Above physiological changes in light of hypertension which is an independent risk factor in precipitating AAA rupture, could result in intra and extra luminal pressure fluctuations and possible rupture.[14] However, our data failed to show any casual relation between hypertensive group of rAAA patients and AP. In fact the number of hypertensive patients showed an almost equal spread in all seasons (Spring $n = 7$, Summer $n = 8$, Autumn $n = 7$, winter $n = 6$)(Table 1.3).

It has also been suggested that smoking which is an independent risk factor for AAA growth, might be related to seasonal and monthly rAAA under the effect of AP alterations.[15,16] Indoor smoking especially in colder months has shown to produce 1.5–6.0 times higher levels of tobacco pollution than the outside environment.[17] Therefore AP changes in conjunction with tobacco induced haemodynamic disturbances might be an explanation for higher suggested rupture incidences in winter months.[18] However, while it is well established that smoking causes damage to the vessel wall, there is no comprehensive explanation as to how active smoking precipitates into rupture and our data failed to exhibit any correlation between smoker group of rAAA and AP (Table 1.3).

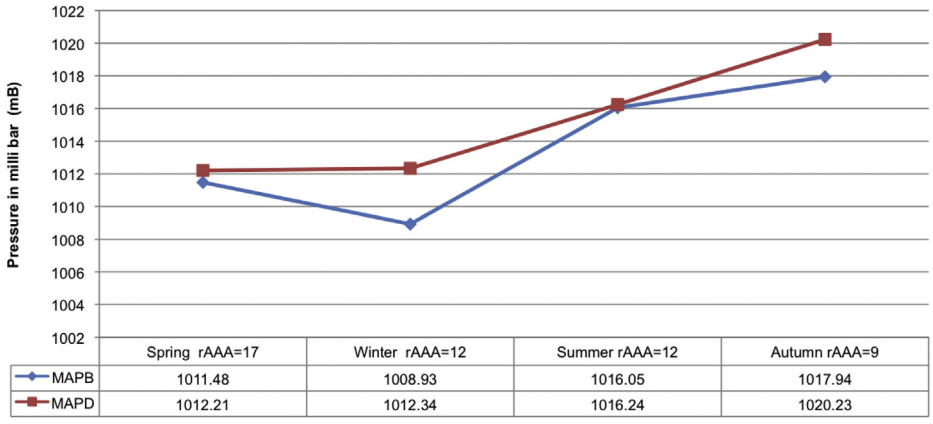
It has been speculated that seasonal variations in rAAA might be due to AP fluctuation (increase Vs decrease) that may indirectly affect the "vessel wall stress" by creating a larger pressure difference across the wall.[19] The ability of the abdominal aorta to withstand this pressure difference is thought to be determined by the strength of the vessel wall and a large pressure difference, may add to the pre-existing deformed aorta and cause rupture.[20,21] Our data showed a possible correlation in both monthly and seasonal AP fluctuations and of rAAA ($p = 0.059$, mean monthly pressure difference = 2.08 mB; range, −2.51–10.85 mB; 95% CI ± 2.93), ($p = 0.054$, mean seasonal pressure difference = 1.65 mB; range, 0.19–3.41 mB; 95% CI ± 0.10)(Graph 1.1) (Graph 1.2).

Talbot et al. were the first to suggest, in their study in 1972, a seasonal peak (winter season) in AAA and thoracic aorta rupture incidence.[22] These findings were complimented by Ballaro et al. who conducted a large research ($n = 19{,}599$) across England and Wales to find a statistically significant higher incidence of rupture during winter season ($p = 0.01$).[23] However, neither of the studies offered any data on AP to determine whether there was a casual link between pressure and rupture ratio. Despite above outcomes our data showed higher incidences of rAAA in spring than winter ($n = 17$; Mean pressure: 1012.21 mB Vs $n = 12$ Mean pressure: 1012.34 mB) although the mean seasonal pressure in both seasons (spring and winter) was similar and much lower than other seasons (summer mean pressure: 1016.24 mB & Autumn mean pressure: 1020.23 mB) (Graph 1.2).



	Jan rAAA=4	Feb rAAA=6	Mar rAAA=7	Apr rAAA=6	May rAAA=4	Jun rAAA=7	Jul rAAA=4	Aug rAAA=1	Sep rAAA=2	Oct rAAA=2	Nov rAAA=5	Dec rAAA=2
MAPB	1017.45	1002.35	1007.47	1010.75	1016.23	1016.8	1012.86	1018.5	1016.3	1016.8	1012.72	1007.15
MAPD	1014.35	1004.68	1015.4	1007.73	1013.52	1015.24	1015.3	1018.2	1023.85	1018.55	1018.31	1018

Graph 1.1. Showing the mean AP on the day (MAPD) and day before (MAPB) and number of ruptures per month, higher AP is associated with lower rAAA ($n = 50$)(2005–2012).



	Spring rAAA=17	Winter rAAA=12	Summer rAAA=12	Autumn rAAA=9
MAPB	1011.48	1008.93	1016.05	1017.94
MAPD	1012.21	1012.34	1016.24	1020.23

Graph 1.2. Showing the mean AP on the day (MAPD) and day before (MAPB) and number of ruptures per season, the drop in rAAA as AP increases ($n = 50$)(2005–2012).

The study by Bown et al. was the first to identify a possible link between AP and rupture incidence. The ten-year long study of 233 patients reaffirmed findings of a peak incidence of AAA rupture in the winter season.[24] It also concluded that there might be a correlation between low AP and rupture ratio that was also noted in our study as the cumulative number of ruptures were much higher in lower AP seasons than those of higher AP (rAAA $n = 29$ at mean atmospheric pressure 1012 mB Vs rAAA $n = 12$ at mean atmospheric pressure 1016 mB Vs rAAA $n = 9$ at mean atmospheric pressure of 1020 mB) (Graph 1.2). The study by smith et al. also showed a possible link between low AP and increased rupture frequencies ($p = 0.025$).[25]

In the category of monthly AP and rAAA, Hakin et al.[26] demonstrated months with lowest mean AP posses higher frequency of rupture ($p < 0.0001$). Similarly, a statistically significant relationship between AP and monthly rupture frequencies was identified ($p < 0.03$). This outcome favours the significant correlation between mean monthly pressures and mean monthly rupture incidence of our data respectively ($n = 12$; $r = -0.61$; $p < 0.034$; rsq = 0.37). Amongst the reviewed literature there was no evidence to demonstrate that the size of the AAA might be related to AP variations and in our study we could not establish any casual link either. Similarly, only two studies could demonstrate a daily correlation of rupture to low AP[25,26] with others failing to assess or show any significant results.

7. Conclusion

The actual cause of infra-renal AAA rupture at any particular period remains elusive to this date. However, there is no doubt that such event is due to multiple number of established factors and physiological alterations. The outcome of this review suggests that periods of low AP irrespective of the day, season or the month are associated with higher incidences of rupture and might need to be considered as an addition to other known precipitating factors. In addition, the evidence gathered might support the policy of prioritisation in elective AAA repair during potentially high-risk seasons (winter and spring) based on association with low atmospheric pressure trends. The actual physiological and haemodynamic impacts of low AP on AAA are currently based on postulated hypothesis that may require further investigation.

Ethical approval

Due to retrospective nature of the study, its lack of intervention in design and revision of the data that was already available for clinical reasons no ethical approval was deemed necessary.

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None.

Author contribution

Ali Kordzadeh designed the study, carried out the analysis , Alan Askari performed the literature search and contributed to the writing of the paper. Mr Panayiotopoulos supervised the paper discussion and final conclusion.

Conflicts of interest

None.

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References

1. Dobust C, Allary M, Oeconomos N. Resection of an aneurysm of the abdominal aorta. Re-establishment of the continuity by a preserved human arterial graft, with result after five months. *AMA Arch Surgery* 1952;**64**:405–8.
2. Nordon IM, Hichlifee RJ, Loftus IM, Thompson IM. Pathophysiology and epidemiology of abdominal aortic aneurysm. *Nat Rev Cardiol* 2011;**8**:92–102.
3. Vardoulaki KA, Walker NM, Day NE, Duffy SW, Ashton HA, Scott RA. Quantifying the risks of hypertension, age, sex and smoking in patients with abdominal aortic aneurysm. *Br J Surg* 2000;**87**(2):195–200.
4. Magalhaes R, Silva MC, Correia M, Bailey T. Are stroke occurrence and outcome related to weather parameters? Results from a population based study in northern portugal. *Cerebrovasc Dis* 2011;**32**(6):542–51.
5. Douglas AS, Allan TM, Rawles JM. Composition of seasonality of disease. *Scott Med* 1991;**36**:76–82.
6. Hademenos GJ, Massoud T, Valentino DJ, Duckwiler G, Viñuela F. A nonlinear mathematical model for the development and rupture of intracranial saccular aneurysms. *Neurol Res* 1994;**16**:376–84.
7. Wixon CL. Vascular hemodynamics. In: Hallet JW, Mills JL, Earnshaw JJ, Reekers JA, Rooke TW, editors. *Comprehensive vascular and endovascular surgery*. 2nd ed. Philadelphia: Mosby Elsevier; 2009. p. 39–44.
8. Bozkurt MA, Temizgonlu KB, Kones O, Alis HA. Rare reason of abdominal compartment syndrome: non-Hodgkin Lymphoma. *J Korean Surg Soc* 2012;**83**:242–5.
9. Burnett RW, Itano M. An interlaboratory study of blood-gas analysis: dependence of pO2 and pCO2 results on atmospheric pressure. *Clin Chem* 1989;**35**(8):1779–81.
10. Woodhouse PR, Khaw KT, Plummer M. Seasonal variation of blood pressure and its relation to ambient temperature in an elderly population. *J Hypertens* 1993;**11**(11):1267–74.
11. Charach G, Rabinonovich PD, Weintraub M. Seasonal changes in blood pressure and frequency of related complications in elderly Israeli patients with essential hypertension. *Gerontology* 2004;**50**(5):315–21.
12. Chapleau MW, Hajduczok G, Abboud FM. Mechanism of resetting of arterial baroreceptors: an overview. *Am J Med Sci* 1988;**295**(4):327–34.
13. Korner PI. Baroreceptor resetting and other determinants of baroreflex properties in hypertension. *Clin Exp Pharmacol Physiol Suppl* 1989;**15**:45–64.

14. Strachan DP. Predictors of death from aortic aneurysm among middle-aged men: the Whitehall study. *Br J Surg* 1991;**78**(4):401–4.
15. Sweeting NH, Bhaskar A, Hunt K. Positive association between consumerism and tobacco and alcohol use in early adolescence: cross sectional study. *BMJ* 2012 sep 24;**2**(5).
16. Ketn KC, Zwolak RM, Egorova NN, Riles TS, Manganaro A, Moskowitz A, et al. Analysis of risk factors for abdominal aortic aneurysm in a cohort of more than 3 million individuals. *J Vasc Surg* 2012;**52**(3):539–48.
17. Siegel M. Involuntary smoking in the restaurant workplace. A review of employee exposure and health effects. *JAMA* 1993;**270**(4):490–3.
18. Chandra S, Chaloupka FJ. Seasonality in cigarette sales: patterns and implications for tobacco control. *Tob Control* 2003;**12**(1):105–7.
19. Stringfellow MM, Lawrence PF, Stringfellow RG. The influence of aorta – aneurysm geometry upon stress in the aneurysm wall. *J Surg Res* 1987;**42**(4): 525–33.
20. Viswanath N, Rodkiewicz CM, Zajac S. On the abdominal aortic aneurysm: pulsatile state consideration. *Med Eng Phys* 1997;**19**(4):343–51.
21. Saul GD. Arterial stress from intraluminal pressure modified by tissue pressure offers a complete explanation for the distribution of atherosclerosis. *Med Hypotheses* 1999;**52**(4):349–51.
22. Talbot S, Langman MJ. Epidemiological features of ruptured dissecting and saccular aortic aneurysms. *Postgrad Med J* 1972;**48**(561):414–6.
23. Ballaro A, Cortina-Borja M, Collin J. A seasonal variation in the incidence of ruptured abdominal aortic aneurysms. *Eur J Vasc Endovasc Surg* 1998;**15**(5): 429–31.
24. Bown M, McCarthy MJ, Bell PR, Sayers RD. Low atmospheric pressure is associated with rupture of abdominal aortic aneurysms. *Eur J Vasc Endovasc Surg* 2003;**25**(1):68–71.
25. Smith RA, Edwards PR, Da Silva AF. Are periods of low atmospheric pressure associated with an increased risk of abdominal aortic aneurysm rupture? *Ann R Coll Surg Engl* 2008;**90**(5):389–93.
26. Harkin DW, O'Donnell M, Butler J, Blair PH, Hood JM, Barros D'Sa AA. Periods of low atmospheric pressure are associated with high abdominal aortic aneurysm rupture rates in Northern Ireland. *Ulster Med* 2005;**74**(2):113–21.